CASE REPORTS

Cardiogenic syncope and epilepsy

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SYNCOPE has been defined as 'a failure of the heart's action resulting in loss of consciousness or sometimes in death' (O.E.D.). Since Gower's discussion of vaso-vagal attacks (1907) many forms of reflex syncope have been described and studied, among them cough syncope (Baker, 1949), carotid sinus syncope (Weiss, 1935) and micturition syncope (Coggins, Lillington & Gray, 1964). The sudden and transient interruption of consciousness seen in a syncopal attack is not uncommonly associated with organic heart disease—cardiogenic syncope (Brigden, 1966)—occurring as it may with sudden changes of rhythm, heart block, paroxysmal tachycardia, myocardial infarction, aortic or pulmonary stenosis, ball-valve obstruction of the mitral valve and lesions associated with severe pulmonary hypertension. The physiological mechanisms involved are reviewed in detail by Wayne (1961). That syncope, if prolonged more than 15-20 sec, may be followed by epileptiform convulsions and incontinence is well attested (Spens, 1793; Campbell, Symonds & Williams, 1950) occurring particularly often in the dentist's chair (Symonds, 1950). The differentiation between epilepsy and syncope in certain cases therefore may be difficult, for although implicit in a diagnosis of epilepsy is a consideration of a primary disorder of brain (Williams, 1958), variants of focal epilepsy, possibly with a hypothalamic excitatory point, may produce apparent syncopal attacks with reflex cardiovascular changes (Kinnier Wilson, 1928; Kershman, 1949). The establishment of a not immediately apparent cardiac cause for syncope in four patients who were referred to a neurological clinic with an initial diagnosis of epilepsy was felt to warrant drawing further attention to this diagnostic problem, particularly as three were seen within the space of 1 month.

Case reports

Case 1

Mr A.W., a 47-year-old clerk, first presented in June 1964 with a 3 months' history of many episodes of transient unconsciousness preceded by 'a feeling spreading from the stomach to the brain'. There were no convulsions or incontinence but he was breathless following attacks.

On examination, there were no abnormalities, the blood pressure was 160/100 mmHg and repeated ECGs and EEGs were normal. A chest X-ray showed an opacity at the left hilum, a Mantoux test was negative at a dilution of 1:100 and bronchoscopy and examination of the sputum for malignant cells was negative. The CSF protein was 74 mg/100 ml and it was thought possible that in view of the normal cardiograms his attacks were of cerebral dysrhythmia associated with cerebral sarcoidosis. He did not respond to anti-convulsant therapy, however, and in October 1964 a transient period of complete heart block was noted in lead aVr of an otherwise normal routine ECG (Fig. 1a). On continuous ECG monitoring it was confirmed that his attacks were synchronous with periods of complete atrio-ventricular block (Fig. 1b). He was treated with ephedrine and

FIG. 1(a)

FIG. 1(b)

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ceased to have attacks of unconsciousness though still having an irregular pulse and atrio-ventricular block on ECG from time to time. Cardiac sarcoidosis as a cause for his arrhythmia has not been proved, a lymph node biopsy being normal.

Case 2

Mr T.P., an introspective 59-year-old compositor, had had 2 years psychotherapy for 'queer feelings mounting up from his stomach' whenever he walked out of his home. In March 1966, however, this symptom complex was followed by sudden loss of consciousness; this recurred 3 weeks later and he was referred to Neurological Out-Patients with a diagnosis of '? epilepsy'. His 26-year-old daughter has confirmed grand mal epilepsy.

On examination, there was no neurological abnormality but a regular pulse rate of 80 alternated with a slow irregular rate of 48. The blood pressure was 180/100 mmHg and cardiac examination was otherwise normal. An ECG showed a

FIG. 2(a)

sinus rhythm with a PR interval of 0.24 sec (Fig. 2a) followed by a variable A-V block, culminating in complete A-V block. This could be precipitated by the Valsalva manoeuvre (Fig. 2b) which

FIG. 2(b)

brought on an attack of transient unconsciousness preceded by his characteristic aura. Investigations revealed no cause for his conduction defect, an EEG was normal, there was no evidence of ischaemic heart disease and he has responded well to long-acting isoprenaline (Saventrine).

Case 3

Mr G.L., a 34-year-old bus driver, was referred urgently to Neurological Out-Patients in June 1966 because in the previous 2 weeks he had had three attacks of unconsciousness immediately preceded by 'a sick feeling in the stomach going up to the head'. He also admitted to short-windedness climbing hills for the last week but had never had pain or constriction in the chest. There was no significant family history.

On examination no abnormality was found but an ECG showed a resolving posterior infarction. He refused admission to hospital and survived convalescence at home and a holiday camp. When seen 4 weeks later he had had no further attacks. An ECG showed considerable resolution, the serum cholesterol was 380 mg/100 ml and the serum beta globulin 1.6 g/100 ml. He has been started on clofibrate ('Atromid S') 250 mg q.d.s. His EEG was normal.

Case 4

Mrs D.L., a 38-year-old housewife, attended Neurological Out-Patients in June 1966 with a history of four attacks of sudden loss of consciousness in the preceding 3 months, two of these being on micturition early in the morning. An 'aura' of ringing in the ears was constantly present just before the attacks but there were no convulsions or incontinence. Her only previous illness was mild hypertension during her second pregnancy 9 years previously, but her mother died of 'heart trouble' aged 46. The only abnormality on physical examination was a blood pressure of 180/120 mmHg but an ECG showed evidence of myocardial infarction. She was admitted and anticoagulated. SGOT estimations were normal as were the WBC and ESR, but serial ECGs confirmed the presence of a postero-lateral myocardial infarct, though its age was difficult to assess. Her convalescence was complicated by a retro-peritoneal bleed which made a blood trans-

fusion necessary, but she was discharged home feeling well 6 weeks after admission. An EEG was normal.

Discussion

Of the four patients described two were established as suffering from intermittent heart block while two had had myocardial infarctions. The presentation of the infarctions was atypical not only in that both were painless but also that they occurred in an age-group in which myocardial infarction is relatively uncommon, though in Case 3 there was hypercholesterolaemia and in Case 4 a maternal family history of heart disease was obtained.

A significant pointer to the diagnosis was found in the patient's own descriptions of the bodily sensations heralding an attack and resembling an 'aura'. Three of the four used almost identical words to describe a radiation upwards from the epigastrium of an odd sensation. Gowers in 1881 similarly described symptoms which he ascribed to vaso-vagal attacks but which may have been due to cardiogenic syncope, while William Bennett in 1827, discussing a patient with a long history of 'epilepsy' in which a slow pulse was noted, recorded 'a sensation as if something arose in the stomach and proceeded upward to the head, followed by all the usual symptoms of epilepsy'.

The electroencephalogram was normal in all of these cases, though an EEG was not obtained during an attack. The EEG in syncope shows generalized slowing with low voltage rather than the characteristic changes of a cerebral dysrhythmia (Kershman, 1949; Gastaut & Fischer-Williams, 1957) but in routine clinical work an EEG during an attack is unlikely to be available, and EEG abnormalities between syncopal attacks have been demonstrated in up to 30% of patients (Williams, 1950). Although the cases recorded did not manifest convulsions or incontinence, these occurrences by no means exclude a diagnosis of cardiogenic syncope. Of the seven cases of unconsciousness produced by heart block reviewed by Sir William Stokes in his original paper (1846) at least three had convulsions (though Robert Adams' case in 1827 did not) and the first recorded case of this condition, by Morgagni in 1769, describes these vividly: 'Anastasio Poggi, a grave and worthy priest in his sixty-eighth year, of habit moderately fat and of a florid complexion, was first seized with epilepsy . . . and left behind slowness of the pulse and in a like manner coldness of the body. Distortions of the eyes, agitations of the limbs and suspensions of all the senses always accompanied the attacks and even now and then an involuntary efflux of urine attended.'

The irregular pulse noted on initial examination of Case 2 was the only physical sign noted in any of the patients which gave a direct lead to the diagnosis. It is suggested from these findings that unsuspected organic heart disease may be present in patients presenting with attacks of loss of consciousness with or without convulsions, that a visceral aura may be described and that the diagnosis of cardiogenic syncope should be considered in such cases. The value of ECG examination in these circumstances is evident.

Summary

Four cases of cardiogenic syncope presenting in a neurological clinic with an initial diagnosis of epilepsy are described. The distinction between epilepsy and syncope and their differential diagnosis are discussed.

Acknowledgments

I am indebted to Dr A. M. G. Campbell for permission to publish details of patients under his care and for his helpful advice and suggestions.

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